The role of interference between vectors in control of plant diseases

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Abstract

Aphids are the primary vector of plant viruses. Transient aphids, which probe several plants per day, are considered to be the principal vectors of non-persistently transmitted (NPT) viruses. However, resident aphids, which can complete their life cycle on a single host and are affected by agronomic practices, can transmit NPT viruses as well. Moreover, they can interfere both directly and indirectly with transient aphids, eventually shaping plant disease dynamics. By mean of an epidemiological model, originally accounting for ecological principles and agronomic practices, we explore the consequences of fertilization and irrigation, pesticide deployment and roguing of infected plants on the spread of viral disease in crops. Our results indicate that the spread of NPT viruses can be *i*) both reduced or increased by fertilization and irrigation, depending on whether the interference is direct or indirect; *ii*) counter-intuitively increased by pesticide application and *iii*) reduced by roguing infected plants. We show that a better understanding of vectors' interactions would enhance our understanding of disease transmission, supporting the development of disease management strategies.

Keywords: aphid-borne pathogen; disease ecology; epidemiological model; inter-specific interference; non-persistently transmitted virus; plant–herbivore interactions; plant–pathogen interactions.

Introduction

Aphids transmit nearly 30% of known plant virus species (Gray and Banerjee, 1999; Brault et al., 2010). Aphids vector the majority of non-persistent transmitted (NPT) viruses with virus particles (virions) remaining loosely attached to the insect's stylets (Ng and Falk, 2006; Brault et al., 2010). According to this transmission mode, virions are rapidly acquired from infected plants, briefly retained by their vector and inoculated to healthy plants during plant sampling probes (Nault, 1997; Ng and Falk, 2006). NPT viruses are responsible for severe damage to crops (Andret-Link and Fuchs, 2005). For instance, *Plum pox virus* (PPV), which is vectored by more than 20 aphid species worldwide, is responsible for sharka, the most devastating disease of stone fruit trees (Rimbaud et al., 2015a). *Potato virus Y* (PVY), which is spread by more than 50 aphid species, threatens the production of a range of solanaceous crops, including potato, tomato, tobacco, and pepper (MacKenzie et al., 2013).

The epidemiology of NPT viruses is closely related to the behaviour of aphid vectors, in particular to *i*) aphids' ability to acquire and inoculate the virus during sampling probes and *ii*) their propensity for moving among plants (Ng and Perry, 2004). With respect to a given plant host species, aphid species can be classified as: "residents", which under favourable conditions, spend most of their life on the same host plant individual, or "transients", which land and probe numerous plant individuals in the same day (Van Emden and Harrington, 2007; Fereres and Moreno, 2009). Although transient aphids are commonly considered the principal vectors of NPT viruses, resi-

dent aphids can also efficiently transmit NPT viruses when they are induced to change their host, for example in response to crowding or to changes in plant nutrient contents (Müller et al., 2001). For instance, the green peach aphid *Myzus persicae*, despite being a resident aphid species that colonises peach *Prunus persica* and potato *Solanum tuberosum* plants (Van Emden and Harrington, 2007; Robert et al., 2000), was observed to be an efficient vector of two NPT viruses (PPV and PVY) at laboratory conditions (Robert et al., 2000; Rimbaud et al., 2015a,b). In addition, the presence of resident aphids may affect transient aphids' behaviour (Kaplan and Denno, 2007; Bird et al., 2019; Chisholm et al., 2019). For example, in their experiment with three aphid species, Mehrparvar et al. (2014) showed that aphid presence on a plant discourages other aphid species to visit the same plant.

Different mechanisms of interference might characterize interactions between resident and transient aphid species. Resident aphids can interfere *i*) directly through the production of pheromones that can have a repelling effect towards other aphid species (Van Emden and Harrington, 2007); or *ii*) indirectly, by inducing the host plant to produce volatile compounds as a defensive mechanism, which may lower plant attractiveness to other aphid species (Zust and Agrawal, 2016). Such interference mechanisms are likely to reduce the number of plants visited by transient aphids in a given area and increase their propensity for leaving the area (Levins and Culver, 1971; Nee and May, 1992).

As far as we know, the effect of the interference between resident and transient aphids on the spread of NPT viruses has never been explored. Understanding NPT viruses spread is complex because experimentation is

costly and difficult: symptoms may be difficult to detect and experimental trials in the vicinity of susceptible commercial crops may be restricted (Cunniffe et al., 2014; Picard et al., 2017). Mathematical models are thus particularly useful to provide complementary insights on virus spread (Jeger, 2020), and to design and test management strategies, while circumventing the difficulties associated with experiments [*e.g.* Fabre et al. (2012); Rimbaud et al. (2015b); Cunniffe and Gilligan (2020)]. Numerous models have been developed to study the role of vector population dynamics and vector-host-pathogen interactions on the spread of NPT viruses [*e.g.* Nakazawa et al. (2012); Sisterson and Stenger (2016); Shaw et al. (2017); Jeger et al. (2018); Allen et al. (2019); Donnelly et al. (2019); Crowder et al. (2019)]. Shaw et al. (2017) developed a model to assess the contributions of vector life history traits (*e.g.* growth rates, fecundity, and longevity) and behavior (*e.g.* vector preferences for settling and feeding) to pathogen spread. Crowder et al. (2019) developed a model where the vector life history traits and behaviour were varied to explore the effect of interaction (*e.g.* predation, competition and mutualism) between a vector and a non vector species on the spread of plant pathogens. However, all existing studies consider only a single vector species per virus, which limits the possibility to assess the effects of interference between two or more vector species.

In the present work, we develop a general epidemiological model which describes the temporal variation of the number of susceptible and infected plants, and of the number of non viruliferous and viruliferous resident and transient aphids in a single field. We apply the model to explore the role of inter-specific interference upon resident and transient aphid behaviour and

the resulting effects on the invasion, persistence and control of NPT viruses in agroecosystems. We use the model to analyze the effects of common agricultural practices, such as fertilization and irrigation, pesticide application and roguing, upon the spread of NPT viruses. We apply the model to a general pathosystem composed of a NPT virus vectored by a resident and a transient aphid species.

Methods

Model outlines and assumptions

The model is schematically represented in Fig.1. The plant population (plant ha^{-1}) is structured into two compartments: susceptible (S) and infected (I). Both resident (subscript "R" hereafter) and transient (subscript "T" hereafter) aphid populations (aphid $plant^{-1}$) are structured into non viruliferous (X_R and X_T) and viruliferous (Z_R and Z_T). The total number of plants per hectare is N_P ($N_P = S + I$), the average number of resident aphids per plant is N_R ($N_R = X_R + Z_R$) and the average number of transient aphids per plant is N_T ($N_T = X_T + Z_T$). A susceptible plant can be infected if it enters in contact with a viruliferous aphid, resident (Z_R) or transient (Z_T). The probability per unit time that a susceptible plant becomes infected (*i.e.* force of infection) depends on i) the rate of contact between plant and aphid $\Lambda_i(X_i + Z_i)$ ($i = R, T$) where Λ_i is the number of plants visited by an aphid per unit time and $(X_i + Z_i)$ is the average number of aphids per plant, ii) the probability $\frac{Z_i}{X_i+Z_i}$ that the contact is indeed with a viruliferous aphid and

iii) the probability δ_i that the contact leads to virus inoculation. Therefore the number of infected plants per unit time at the plant population scale is $\sum_{i=R,T} \Lambda_i \delta_i Z_i S$.

We assumed that infected plants are removed at a *per capita* rate ρ and that both susceptible and infected plants are harvested at a *per capita* rate θ. Furthermore, Γ new susceptible plants are planted per unit time. A non viruliferous aphid can become viruliferous if it enters in contact with an infected plant. The probability per unit time that a non viruliferous aphid acquires the virus from an infected plant depends on *i*) the rate of contact between one aphid and a plant Λ_i, *ii*) the probability $\frac{I}{N_P}$ that the contact is indeed with an infected plant and *iii*) the probability ε_i that the contact leads to virus acquisition. Viruliferous aphids lose viruliferousness at a clearance rate γ. We assume that the population of resident aphids varies following a logistic function with a density dependent *per capita* birth rate $r(1 - \frac{N_r}{h})$, where r is the intrinsic growth rate and h is the plant hosting capacity with respect to aphid population (*i.e.* the resident aphid population size at which birth rate is zero), and a constant aphid mortality rate μ. Resident aphids can leave their host plant and move to another one as a response to unfavourable environmental conditions (Müller et al., 2001): we assumed $\Lambda_T > \Lambda_R > 0$, due to the different moving behaviour of transient and resident aphids. We assume that an average of λ transient aphids per plant immigrate into the system (*i.e.* a patch of 1 ha) per unit time and a fraction π of them are viruliferous. Such a fraction depends on the disease prevalence in the surrounding area. Transient aphids emigrate from the system or die at a rate τ, which is the inverse of their average sojourn time in the system.

We assume that resident aphids exert two types of interference towards transient aphids, "visiting" and "emigration" interference, that will be presented in the following sections.

Model equations

The model outlined above can be described by the following set of differential equations:

$$\begin{cases} \dot{S} = \Gamma - (\Lambda_R \delta_R Z_R + \Lambda_T \delta_T f(\cdot) Z_T) S - \theta S \\ \dot{I} = (\Lambda_R \delta_R Z_R + \Lambda_T \delta_T f(\cdot) Z_T) S - (\rho + \theta) I \\ \dot{X_R} = r N_R (1 - \frac{N_R}{h}) - \mu X_R - \Lambda_R \varepsilon_R \frac{I}{N_P} X_R + \gamma Z_R \\ \dot{Z_R} = \Lambda_R \varepsilon_R \frac{I}{N_P} X_R - (\gamma + \mu) Z_R \\ \dot{X_T} = (1 - \pi)\lambda - \Lambda_T \varepsilon_T f(\cdot) \frac{I}{N_P} X_T + \gamma Z_T - \tau g(\cdot) X_T \\ \dot{Z_T} = \pi\lambda + \Lambda_T \varepsilon_T f(\cdot) \frac{I}{N_P} X_T - \gamma Z_T - \tau g(\cdot) Z_T \end{cases} \quad (1)$$

Where the dot represents the derivative with respect to time t, and parameters are as in Table 1. Functions $f(\cdot)$ and $g(\cdot)$ represent, respectively, "visiting interference" and "emigration interference" exerted by resident aphids towards transient aphids. Details on their functional forms are given in the following section.

The set of differential equations can be used to represent a plant virus epidemic under very general circumstances. To gain insight on disease transmission, we assume that the total number of plants (N_P) is constant (*i.e.* every harvested or rogued plant is immediately replaced), which implies that

the number Γ of new susceptible plant planted per unit time is given by

$$\Gamma = \theta S + (\rho + \theta) I \tag{2}$$

where θ is plant harvesting rate and ρ is infected plant roguing rate. Moreover, the total number of transient aphids per plant in absence of resident aphids (T) is constant, which implies that the average number λ of transient aphids immigrating into the system per plant per unit time is given by

$$\lambda = \tau T \tag{3}$$

Modelling interference between resident and transient aphids

We assume that interference exerted by resident towards transient aphids can independently induce them to: *i*) visit fewer plants per unit time (visiting interference); and/or *ii*) reduce the average sojourn time in the system (emigration interference). Moreover, for both visiting and emigration interference, we consider two interference scenarios:

- direct [*e.g.* competition for space, (Van Emden and Harrington, 2007)], where interference depends upon the density of resident aphids on the host ($\frac{N_R}{h}$). This implies that, at the same aphid abundance (N_R), the exerted interference is weaker on a bigger plant (*i.e.* higher plant hosting capacity h).

- Indirect [*e.g.* release of plant volatiles, (Zust and Agrawal, 2016)],

where interference depends upon the absolute number of resident aphids and it is independent from the plant hosting capacity.

Agricultural practices, such as fertilization and irrigation, that influence plant size, may have different effects on plant virus epidemics according to which interference scenario is under consideration. Forms of interference intermediate between these two extreme scenarios are possible and they can be considered in our model, as presented in the *Supplementary Information*.

Visiting interference

Visiting interference controls the proportionate decrease in the rate at which transient aphids visit plants, via two functional forms $f(\frac{N_R}{h})$ and $f(N_R)$, respectively for direct and indirect interference scenarios :

$$f\left(\frac{N_R}{h}\right) = \frac{1}{1+\left(\nu_1 \frac{N_R}{h}\right)^{\alpha_1}} \qquad f(N_R) = \frac{1}{1+\left(\frac{\nu_1}{h_R} N_R\right)^{\alpha_1}} \tag{4}$$

These are a generalisation of the competition function proposed by Bellows (1981), extending in continuous time the model of Smith and Slatkin (1972). They are sufficiently flexible to account for a range of possible types of interference (Fig. 2). The "strength" parameter ν_1 controls the magnitude of interference, and so the density of residents that is required to appreciably affect the behaviour of transient aphids. In our model the value of ν_1 is defined in reference to the direct interference scenario. To assure that its value is biologically relevant also in the indirect interference scenario, we scale it with the "reference" value of plant hosting capacity (h_R). This implies that, when $h = h_R$ - all other things being equal (*i.e.* the values of α_1 and N_R) - the

value of the interference function is the same independently from the underlying interference scenario. The "curvature" parameter α_1, controls whether the visiting function presents an inflection point (for $\alpha_1 > 1$) or not (for $0 < \alpha_1 \leq 1$).

Emigration interference

Emigration interference controls the proportionate increase in the rate at which transient aphids leave the system, via two functional form $g(\frac{N_R}{h})$ and $g(N_R)$, respectively for direct and indirect interference scenarios:

$$g\left(\frac{N_R}{h}\right) = 1 + \left(\nu_2 \frac{N_R}{h}\right)^{\alpha_2} \qquad g(N_R) = 1 + \left(\frac{\nu_2}{h_R} N_R\right)^{\alpha_2} \tag{5}$$

The emigration interference function is characterized by two parameters determining strength and curvature (ν_2 and α_2), and, in case of indirect interference scenario, also by the parameter h_R. All three parameters have identical interpretations to those controlling the visiting interference. In the most general form of our model, the analogous parameters for the two functions are entirely independent (*e.g.* $\alpha_1 \neq \alpha_2$), which provides sufficient flexibility to allow inter-specific interference to affect visiting and/or emigration independently. However in all numerical work in this paper we assumed that $\nu_1 = \nu_2 = \nu$ and $\alpha_1 = \alpha_2 = \alpha$, and so, in turn, that proportionate effects of the density of resident aphids on visiting and emigration rate were similar.

Agricultural practices and disease control

A number of agricultural practices are commonly used to *i*) increase plant growth, *ii*) control aphid populations and *iii*) mitigate the effect of plant diseases. The effect of these practices can be taken into account by modifying the values of some model parameters from their reference value. Practices such as fertilization or irrigation, commonly used to foster crop growth, may increase the abundance of resident aphids dwelling on the plants (Huberty and Denno, 2006; Tamburini et al., 2018) and in our model this translates into an increase of the plant hosting capacity h (while parameter h_R is not varied). Pesticides are commonly used in crops to reduce the number of resident aphids and in our model this translates into an increase of resident aphid mortality μ, while the dynamics of transient aphids is not affected (Perring et al., 1999). In case of spread of plant diseases, frequently producers try to identify as soon as possible the infected plants and replace them with new healthy plants, a practice known as roguing (Sisterson and Stenger, 2013). To represent the discrete time process of roguing in our continuous time model, we set the rate of removal of infected host plants (ρ) such that the average period for which plants are symptomatically infected is equal to one half of the interval between successive rounds of roguing (Cunniffe et al., 2014).

Methods of analysis

We first determine the system equilibria and the basic reproduction number of the disease, R_0. Then, we explore the responses of R_0 to variations of

the aforementioned agricultural practices (modeled through parameters h, μ and ρ) under the direct and indirect interference scenarios. In particular, we vary one parameter at a time and we explore the effectiveness of the respective agricultural practices in controlling and eradicating the disease. Finally, we analyse the effectiveness of combinations of agricultural practices in controlling and eradicating the disease by exploring the response of R_0 to simultaneous variation of two parameters at a time. In our analysis, we use biologically plausible parameter values and ranges to reflect a broad range of single host - multi vector system rather than restricting the analysis to a specific system.

Results

Equilibrium analysis

The behaviour of the model at equilibrium is summarized in Table 2. When $\mu \geq r$ (*i.e.* the mortality rate of resident aphids is larger than their intrinsic growth rate), resident aphids are not able to survive ($\bar{X}_R = \bar{Z}_R = 0$) and the disease is spread exclusively by transient aphids. When $\mu < r$ both resident and transient aphids are present in the system and may spread the disease. When all the incoming transient aphids are non viruliferous (*i.e.* $\pi = 0$), the disease is able to persist only if the basic reproduction number (R_0, presented in the following section) is higher than 1, otherwise the disease is eradicated (Diekmann et al., 1990). When a fraction of incoming transient aphids is viruliferous ($\pi > 0$), the disease is always able to persist,

regardless of whether the basic reproduction number is smaller or larger than 1, because there will always be an influx of some new viruliferous aphids into the system, and infections of plants will result not just from infected plants in the system, but also from viruliferous individuals originating from outside the system [similar to Madden et al. (2000)]. The patterns presented in Table 2 can be explained analytically, with the mathematical details derived in the *Supplementary Information*.

The basic reproduction number

In our system, when there is no immigration of viruliferous aphids ($\pi = 0$), the basic reproduction number R_0 is expressed as

$$R_0 = \frac{1}{\rho + \theta}\left(\frac{\Lambda_R^2 \delta_R \varepsilon_R \bar{N}_R}{\gamma + \mu} + \frac{\Lambda_T^2 \delta_T \varepsilon_T f(\cdot)^2 \bar{N}_T}{\gamma + \tau g(\cdot)}\right) \tag{6}$$

and it can be written as the sum of two components, $R_0 = R_0^R + R_0^T$, where $R_0^R = \frac{\Lambda_R^2 \delta_R \mathcal{E}_R \bar{N}_R}{(\rho+\theta)(\gamma+\mu)}$ and $R_0^T = \frac{\Lambda_T^2 \delta_T \mathcal{E}_T f(\cdot)^2 \bar{N}_T}{(\rho+\theta)(\gamma+\tau g(\cdot))}$ (see the *Supplementary Information* for further details). The first component accounts for disease transmission by resident aphids and the second by transient aphids. Such a representation of the basic reproduction number is typical for plant disease models with multiple routes of transmission (Jeger et al., 2009; Cunniffe and Gilligan, 2010; Cunniffe et al., 2012).

In the equation of the basic reproduction number, the term $(\rho + \theta)^{-1}$ indicates the average time spent by a plant as infected, before it is rogued or harvested; the terms $(\gamma + \mu)^{-1}$ and $(\gamma + \tau g(\cdot))^{-1}$ indicate the average time spent by a viruliferous aphid, respectively resident or transient, in the

system. The terms $\Lambda_R \delta_R \bar{N}_R$ and $\Lambda_T \delta_T f(\cdot) \bar{N}_T$ represent the rates at which a susceptible plant is infected by resident or transient aphids, respectively. The terms $\bar{N}_R$ and $\bar{N}_T$ indicate the number of, respectively, resident and transient aphids per plant at the steady state. The terms $\Lambda_R \varepsilon_R$ and $\Lambda_T \varepsilon_T f(\cdot)$ are the rates at which a resident or a transient aphid acquires the virus from an infected plant, respectively. The terms $f(\cdot)$ and $g(\cdot)$ are, respectively, the function for visiting and emigration interference evaluated at the steady state, for the direct interference scenario $[f(\cdot) = f(\frac{\bar{N}_R}{h})$ and $g(\cdot) = g(\frac{\bar{N}_R}{h})]$ and for the indirect interference scenarios $(f(\cdot) = f(\bar{N}_R)$ and $g(\cdot) = g(\bar{N}_R)]$.

The size of the resident aphid population ($\bar{N}_R$) appears both at the numerator of the R_0^R component and as an argument of the interference functions $f(\cdot)$ and $g(\cdot)$ which respectively decrease and increase with $\bar{N}_R$. This implies that the resident component, R_0^R, of the basic reproduction number increases with $\bar{N}_R$ while the transient component, R_0^T, decreases with it. According to the assumed population dynamics of resident and transient aphids, $\bar{N}_R = h(1 - \mu/r)$ if $\mu < r$, $\bar{N}_R = 0$ if $\mu \geq r$ and $\bar{N}_T = \lambda/(\tau g(\cdot))$ (see the *Supplementary Information* for further details). This suggests that the response of the basic reproduction number to h, μ and r might be non-monotonic.

Note that, whenever the basic reproduction number is higher than 1, the disease is not eradicated and the incidence of virus infection in plant can be computed as $\frac{\bar{I}}{N_P}$, where $\bar{I}$ is the size of infected plant population at equilibrium and N_P is the total number of plants in the system (assumed constant). The response of the incidence of virus infection in plants to variation of parameters h, μ and ρ correlates strongly with that of R_0 (see Fig. 2 in the *Supplementary Information*).

The role of plant hosting capacity

The response of the basic reproduction number to aphid hosting capacity is summarized in Fig. 3A. When the system conditions can sustain a population of resident aphids (*i.e.* $r > \mu$), its equilibrium value increases with the plant hosting capacity h. This always translates into an increase of the term R_0^R. On the other hand, the effect of increasing h on the term R_0^T is mediated by the interference functions, $f(\cdot)$ and $g(\cdot)$. Such an effect is null when the interference is direct (see equations 4 and 5, for $\bar{N}_R = h(1 - \mu/r)$) and it is negative when the interference between aphids is indirect. Increasing plant size, and consequently its hosting capacity, is followed by an increase of the population of resident aphids dwelling on a plant. This has no effect on the transient aphid population when the interference is direct, because the density of resident aphids is kept constant, but it negatively affects the transient aphid population when the interference is indirect. When the interference is direct, increasing the value of h increases the basic reproduction number but, in the presence of indirect interference the response of R_0 is non-monotone, with a minimum value obtained for intermediate values of hosting capacity.

The role of resident aphid mortality

The effect of aphid mortality (μ) on the basic reproduction number is summarized in Fig. 3B. Increasing μ has a negative effect on the R_0^R term reducing both the resident population density and the average time spent by a viruliferous resident aphid in the system. On the other hand, it has a positive effect on the R_0^T term by releasing interference forces exerted by resident

aphids and consequently increasing transient aphids movement and sojourn time in the system. In this case, the interference scenario (whether direct or indirect) has no influence because the plant hosting capacity is considered at its reference value $h = h_R$ (see equations 4 and 5, for $h = h_R$). Also in this case, a minimum value of R_0 is obtained for intermediate values of resident aphid mortality.

The role of roguing

An increase to the roguing rate ρ decreases the basic reproduction number as it reduces the time that an infected plant spends in the system before it is rogued (see equation 6 and Fig. 3C). As before, the interference scenario has no influence on the value of R_0 because the plant hosting capacity is considered at its reference value $h = h_R$.

Agricultural practices and disease control

The response of R_0 to the simultaneous variation of two control parameters is summarize in Fig. 4. It is always possible to eradicate the disease for some combination of two of the considered parameters. Eradication is possible without the addition of pesticides ($\mu = 0.04$ day^{-1}, *i.e.* resident aphid natural mortality) for relatively small plant hosting capacity (h, which increases with fertilization and irrigation) (Fig. 4A-B). Eradication is not possible for relative high values of resident aphid mortality (μ), independently from the value of h, in the direct interference scenario (Fig. 4B), while it is possible in the indirect interference scenario, where the value of μ that leads to disease

eradication, increases with the value of h (Fig. 4A). Increasing roguing rate (ρ) increases the range of h and μ values that lead to disease eradication. Both for direct and indirect interference scenarios, if infected plant are identified and eliminated every periods of a maximum of 50 days (*i.e.* $\rho \geq 0.04$ day^{-1}), disease eradication is possible for nearly all the considered h values (Fig. 4 C-D) and for nearly all the considered μ values (Fig. 4E).

Discussion

Meta-analytical evaluations (Bird et al., 2019; Kaplan and Denno, 2007) suggests that both direct (*i.e.* aphid mediated) and indirect (*i.e.* plant mediated) interferences between herbivorous insects shape their behaviour and performance. For example, in an experimental work, Mehrparvar et al. (2014) showed that interference between different aphids species affect host selection behaviour, with alate aphid individuals rarely choosing a plant occupied by individuals of another aphids' species. However, these interference mechanisms have been ignored in epidemiological analyses despite a few exceptions [*e.g.* Crowder et al. (2019); Chisholm et al. (2019); Thaler et al. (2010)]. Yet, to our knowledge, the existing experimental and theoretical works only consider interferences between vector and non-vector insects. For example, Chisholm et al. (2019) observed higher rates of *Pea enation mosaic virus* spread when the vector *Acythosiphon pisum* individuals shared hosts with a non vector herbivore *Sitona lineatus*.

In the present work, we modeled the interaction between two aphid vectors via two interference functions, which account for the reduction of the

number of plants visited per unit time and the average resident time in the system of transient aphids. Both direct and indirect interference scenario can be represented depending on the considered pathosystem. By means of mathematical and numerical analyses of our model, we have demonstrated that interference can have profound effects on the invasion, persistence and control of plant NPT viruses. In theoretical work, ecological interactions that alter vector movement limiting the number of plants visited by a vector, have already been shown to be more effective in controlling the spread of NPT viruses than those targeting vector abundance (Crowder et al., 2019). Indeed, when the length of time an infectious vector spends moving, searching for a new host to visit, is long in comparison to pathogen retention time, the pathogen may be cleared in transit before visiting a new susceptible host (Ng and Falk, 2006; Killiny and Almeida, 2014). The theoretical analysis of our model confirm these findings: visiting interference $f(\cdot)$, affecting the number of plants visited by transient aphids, reduces both the probability of acquiring and inoculating the virus. That is why, no matter whether the underlying mechanism is direct or indirect, visiting interference appears squared in the basic reproduction number, which determines a greater effect of the visiting interference in diminishing the invasion and persistence of the disease respect to emigration interference.

Our results suggest that commonly used agricultural practices, such as fertilization and irrigation, pesticide application and roguing of diseased plants, can have unexpected results upon the spread of NPT viruses. Fertilization and irrigation are commonly used in agriculture to meet plants' nutrient and water needs and increase plant growth and production (Gliessman,

2015). Yet they can impact disease development and spread, possibly affecting plant physiology, pathogens and/or vector population dynamics (Dordas, 2008; Miller et al., 2015). On the one hand nitrogen is involved in the resistance mechanisms of the host plant, *i.e.* its ability to limit the development and reproduction of the invading pathogen, possibly decreasing the incidence of disease in crop plants (Dordas, 2008). On the other hand, fertilization and irrigation can increase vector populations via changes in plant nutrient and irrigation status, potentially impacting the spread of plant diseases. For example, the growth rate of wheat curl mite, vector of the *Wheat streak mosaic virus*, was observed to increase with fertilization on winter wheat (Miller et al., 2015). Populations of bird cherry-oat aphid (*Rhopalosiphum padi L.*), vector of the *Barley yellow dwarf virus*, have been observed to increase with irrigation (Hale et al., 2003) and fertilization (Liang et al., 2019) on different grass species. Our results show that an intermediate plant size, which sustains a population of resident aphids large enough to appreciably reduce the spread of the virus by transient aphids, but not too large to prevent disease spread by resident vectors, may lead to disease eradication.

Pesticide application is the most common aphid control method, but it is well known that its ability to prevent the spread of NPT viruses by transient aphids is limited because inoculation occurs rapidly and before a pesticide can take effect on the transient vector (Perring et al., 1999). Furthermore pesticide may affect local pest community structure as differential susceptibility to pesticide may result in species dominance shift favoring secondary pest outbreaks (Mohammed et al., 2019; Zhao et al., 2017). In their experiments with two aphids species, *Rhopalosiphum padi* and *Sitobion avenae*,

Mohammed et al. (2019) showed that pesticide exposure led to a shift in the outcome of interspecific competition between the two aphid species, compromising the dominance of *R. padi* in pesticide-free plants, while favouring the prevalence of the *S. avenae* under pesticides exposure. Our results show that small pesticide application has the potential to slightly reduce the spread of NPT viruses. However, large pesticide application, reducing the interference exerted by resident towards transient aphids, could be counter productive in reducing NPT viruses, because it favours the prevalence of transient aphids, increasing the spread of the virus by the more mobile vector.

Roguing infected plants has often been implemented to control the spread of plant pathogens (Sisterson and Stenger, 2013; Rimbaud et al., 2015a). The success of roguing in slowing disease spread depends on how rapidly infected plants are identified and removed (Cunniffe et al., 2015; Fabre et al., 2021). Yet, there are various logistical issues associated with identification and removal of infected plants in large-scale agriculture. Firstly, the identification of diseased plants may be hampered by a lack of appropriate and/or cost-effective diagnostic tests. Further, growers can be reluctant to remove diseased plants as soon as symptoms are identified, since infected plants may continue to produce a marketable yield (Sisterson and Stenger, 2013). Finally, the degree of coordination among farmers concerning the decision of roguing is likely to affect the success in slowing disease spread (Laranjeira et al., 2020). Our results unsurprisingly suggest that the incidence of the disease decreases with the effort put into roguing. Yet, they would benefit from further economic evaluations, given the cost of roguing and replanting operations.

Despite our efforts to provide a realistic representation of the complex epidemiological and ecological components of an agroecosystem, we had to introduce a number of simplifying assumptions. We have assumed the resident aphid mortality rate to be constant, in accordance to several authors (Crowder et al., 2019; Donnelly et al., 2019; Allen et al., 2019). In reality, the effects of chemical control on pest mortality do not remain constant, but vary with the repeated application and subsequent decay of pesticides' concentration. We have assumed that NPT viruses do not manipulate host-vector behavior in order to enhance their own transmission, for example by making infected plant more attractive to aphids but inhibiting aphid settling on infected plants (Mauck et al., 2012; Donnelly et al., 2019). This may not always hold, for example, it was shown that squash plants (*Cucurbita pepo*) infected with *Cucumber mosaic virus* firstly emit a blend of volatile organic compounds that attracts aphids, and secondly produce antifeedant compound, which deter aphids from prolonged feeding (Pickett and Khan, 2016). Yet, a non negligible number of pathosystems involving viruses considered to be of the NPT transmission type do not follow this "attract and deter" trend (Mauck et al., 2012). Finally, it is possible that plants put in place other types of defensive mechanisms which may impair resident aphid fecundity (Zust and Agrawal, 2016) and which can be fostered by fertilization (Zaffaroni et al., 2020). Although all these mechanisms could be included in our model, we have chosen to avoid the proliferation of parameters which would have been associated with more complex models, possibly hiding the underlying message of this work. Yet, despite the simplifying assumptions outlined above and noting that further experimental works are

clearly required to confirm our findings, our work suggests that the impacts of inter-specific interference should be incorporated more broadly into the planning of disease management strategies for the control and eradication of aphid vectored NPT viruses.

Statement of authorship: MZ, LR, NJC and DB conceived the ideas. MZ, NJC and DB designed the methodology and produced the results. All authors discussed the results and contributed to the writing and editing of the manuscript.

Data accessibility statement: Data sharing is not applicable to this article as no new data were created or analyzed in this study.

References

Allen, L. J., Bokil, V. A., Cunniffe, N. J., Hamelin, F. M., Hilker, F. M., and Jeger, M. J. (2019). Modelling vector transmission and epidemiology of co-infecting plant viruses. *Viruses*, 11(12):1–25.

Andret-Link, P. and Fuchs, M. (2005). Transmission Specificity of Plant Viruses by Vectors. *Journal of Plant Pathology*, 87(3):153–165.

Bellows, T. S. (1981). The Descriptive Properties of Some Models for Density Dependence. *The Journal of Animal Ecology*, 50(1):139–156.

Bird, G., Kaczvinsky, C., Wilson, A., and Hardy, N. (2019). When do herbivorous insects compete? A phylogenetic meta-analysis. *Ecology Letters*, 22(5):875–883.

Brault, V., Uzest, M., Monsion, B., Jacquot, E., and Blanc, S. (2010). Aphids as transport devices for plant viruses. *Comptes Rendus - Biologies*, 333(6-7):524–538.

Chisholm, P. J., Eigenbrode, S. D., Clark, R. E., Basu, S., and Crowder, D. W. (2019). Plant-mediated interactions between a vector and a non-vector herbivore promote the spread of a plant virus. *Proceedings of the Royal Society B: Biological Sciences*, 286(1911).

Crowder, D. W., Li, J., Borer, E. T., Finke, D. L., Sharon, R., Pattemore, D. E., and Medlock, J. (2019). Species interactions affect the spread of vector-borne plant pathogens independent of transmission mode. *Ecology*, 100(9):1–10.

Cunniffe, N. J. and Gilligan, C. A. (2010). Invasion, persistence and control in epidemic models for plant pathogens: the effect of host demography. *Journal of The Royal Society Interface*, 7(44):439–451.

Cunniffe, N. J. and Gilligan, C. A. (2020). Use of mathematical models to predict epidemics and to optimise disease detection and management. In Ristaino, J. B. and Records, A., editors, *Emerging Plant Diseases and Global Food Security*, chapter 12, pages 239–266. APS Press.

Cunniffe, N. J., Koskella, B., E. Metcalf, C. J., Parnell, S., Gottwald, T. R., and Gilligan, C. A. (2015). Thirteen challenges in modelling plant diseases. *Epidemics*, 10:6–10.

Cunniffe, N. J., Laranjeira, F., Neri, F., DeSimone, R., and Gilligan, C. (2014). Cost-Effective Control of Plant Disease When Epidemiological

513 Knowledge Is Incomplete: Modelling Bahia Bark Scaling of Citrus. *PLoS*
514 *Computational Biology*, 10(8).

515 Cunniffe, N. J., Stutt, R. O. J. H., van den Bosch, F., and Gilligan, C. A.
516 (2012). Time-Dependent Infectivity and Flexible Latent and Infectious
517 Periods in Compartmental Models of Plant Disease. *Phytopathology*,
518 102(4):365–380.

519 Diekmann, O., Heesterbeek, J. A., and Metz, J. A. (1990). On the definition
520 and the computation of the basic reproduction ratio R0 in models for
521 infectious diseases in heterogeneous populations. *Journal of Mathematical*
522 *Biology*, 28(4):365–382.

523 Donnelly, R., Cunniffe, N. J., Carr, J. P., and Gilligan, C. A. (2019).
524 Pathogenic modification of plants enhances long-distance dispersal of non-
525 persistently transmitted viruses to new hosts. *Ecology*, 100(7).

526 Dordas, C. (2008). Role of nutrients in controlling plant diseases in sustain-
527 able agriculture. A review. *Agron. Sustain. Dev.*, 28:33–46.

528 Fabre, F., Coville, J., and Cunniffe, N. J. (2021). Optimising reactive disease
529 management using spatially explicit models at the landscape scale. In
530 Scott, P., Strange, R., Korsten, L., and Gullino, M. L., editors, *Plant*
531 *Diseases and Food Security in the 21st Century*, chapter 4, pages 47–72.
532 Springer Nature Switzerland AG.

533 Fabre, F., Rousseau, E., Mailleret, L., and Moury, B. (2012). Durable strate-
534 gies to deploy plant resistance in agricultural landscapes. *New Phytologist*,
535 193(4):1064–1075.

Fereres, A. and Moreno, A. (2009). Behavioural aspects influencing plant virus transmission by homopteran insects. *Virus Research*, 141(2):158–168.

Gliessman, S. R. (2015). *Agroecology: The Ecology of Sustainable Food Systems*. CRC Press, Boca Raton, USA, third edition.

Gray, S. M. and Banerjee, N. (1999). Mechanisms of arthropod transmission of plant and animal viruses. *Microbiology and molecular biology reviews*, 63(1):128–148.

Hale, B. K., Bale, J. S., Pritchard, J., Masters, G. J., and Brown, V. K. (2003). Effects of host plant drought stress on the performance of the bird cherry-oat aphid, *Rhopalosiphum padi* (L.): A mechanistic analysis. *Ecological Entomology*, 28(6):666–677.

Holt, J., Jeger, M. J., Thresh, J., and Otim-Nape, G. W. (1997). An Epidemilogical Model Incorporating Vector Population Dynamics Applied to African Cassava Mosaic Virus Disease. *Journal of applied ecology*, 34(3):793–806.

Huberty, A. F. and Denno, R. F. (2006). Consequences of nitrogen and phosphorus limitation for the performance of two planthoppers with divergent life-history strategies. *Oecologia*, 149(3):444–455.

Jeger, M. J. (2020). The epidemiology of plant virus disease: Towards a new synthesis. *Plants*, 9(12):1768.

Jeger, M. J., Holt, J., Van Den Bosch, F., and Madden, L. V. (2004). Epidemiology of insect-transmitted plant viruses: Modelling disease dynamics and control interventions. *Physiological Entomology*, 29(3):291–304.

Jeger, M. J., Jeffries, P., Elad, Y., and Xu, X. M. (2009). A generic theoretical model for biological control of foliar plant diseases. *Journal of Theoretical Biology*, 256(2):201–214.

Jeger, M. J., Lamour, A., Gilligan, C. A., and Otten, W. (2008). A fungal growth model fitted to carbon-limited dynamics of Rhizoctonia solani. *New Phytologist*, 178(3):625–633.

Jeger, M. J., Madden, L. V., and Van Den Bosch, F. (2018). Plant virus epidemiology: Applications and prospects for mathematical modeling and analysis to improve understanding and disease control. *Plant Disease*, 102(5):837–854.

Jeger, M. J., Van Den Bosch, F., Madden, L. V., and Holt, J. (1998). A model for analysing plant-virus transmission characteristics and epidemic development. *Mathematical Medicine and Biology: A Journal of the IMA*, 15(1):1–18.

Kaplan, I. and Denno, R. F. (2007). Interspecific interactions in phytophagous insects revisited: A quantitative assessment of competition theory. *Ecology Letters*, 10(10):977–994.

Killiny, N. and Almeida, R. P. (2014). Factors affecting the initial adhesion and retention of the plant pathogen xylella fastidiosa in the foregut of an insect vector. *Applied and environmental microbiology*, 80(1):420–426.

Laranjeira, F. F., Silva, S. X. B., Murray-Watson, R. E., Soares, A. C. F., Santos-Filho, H. P., and Cunniffe, N. J. (2020). Spatiotemporal dynamics and modelling support the case for area-wide management of citrus greasy spot in a Brazilian smallholder farming region. *Plant Pathology*, 69(3):467–483.

Levins, R. and Culver, D. (1971). Regional Coexistence of Species and Competition between Rare Species. *Proceedings of the National Academy of Sciences*, 68(6):1246–1248.

Liang, X., Rashidi, M., Rogers, C. W., Marshall, J. M., Price, W. J., and Rashed, A. (2019). Winter wheat (Triticum aestivum) response to Barley yellow dwarf virus at various nitrogen application rates in the presence and absence of its aphid vector, Rhopalosiphum padi. *Entomologia Experimentalis et Applicata*, 167(2):98–107.

MacKenzie, T. D., Fageria, M. S., Nie, X., and Singh, M. (2013). Effects of crop management practices on current-season spread of Potato virus Y. *Plant Disease*, 98(2):213–222.

Madden, L. V., Jeger, M. J., and van den Bosch, F. (2000). A Theoretical Assessment of the Effects of Vector-Virus Transmission Mechanism on Plant Virus Disease Epidemics. *Phytopathology*, 90(6):576–594.

Mauck, K., Bosque-Pérez, N. A., Eigenbrode, S. D., De Moraes, C. M., and Mescher, M. C. (2012). Transmission mechanisms shape pathogen effects on host–vector interactions: evidence from plant viruses. *Functional Ecology*, 26(5):1162–1175.

603 Mehrparvar, M., Mansouri, S. M., and Weisser, W. W. (2014). Mechanisms of
604 species-sorting: Effect of habitat occupancy on aphids' host plant selection.
605 *Ecological Entomology*, 39(3):281–289.

606 Miller, Z. J., Lehnhoff, E. A., Menalled, F. D., and Burrows, M. (2015). Ef-
607 fects of soil nitrogen and atmospheric carbon dioxide on Wheat streak
608 mosaic virus and its vector (Aceria tosichella Kiefer). *Plant Disease*,
609 99(12):1803–1807.

610 Mohammed, A. A. A. H., Desneux, N., Monticelli, L. S., Fan, Y., Shi,
611 X., Guedes, R. N. C., and Gao, X. (2019). Potential for insecticide-
612 mediated shift in ecological dominance between two competing aphid
613 species. *Chemosphere*, 226:651–658.

614 Müller, C. B., Williams, I. S., and Hardie, J. (2001). The role of nutri-
615 tion, crowding and interspecific interactions in the development of winged
616 aphids. *Ecological Entomology*, 26(3):330–340.

617 Nakazawa, T., Yamanaka, T., and Urano, S. (2012). Model analysis for
618 plant disease dynamics co-mediated by herbivory and herbivore-borne phy-
619 topathogens. *Biology Letters*, 8(4):685–688.

620 Nault, L. (1997). Arthropod transmission of plant viruses. *Annals of the*
621 *Entomological Society of America*, 90(5):521–541.

622 Nee, S. and May, R. (1992). Dynamics of Metapopulations : Habitat Destruc-
623 tion and Competitive Coexistence. *Journal of Animal Ecology*, 61(1):37–
624 40.

Ng, J. C. K. and Falk, B. W. (2006). Virus-Vector Interactions Mediating Nonpersistent and Semipersistent Transmission of Plant Viruses. *Annual Review of Phytopathology*, 44(1):183–212.

Ng, J. C. K. and Perry, K. L. (2004). Transmission of plant viruses by aphid vectors. *Molecular Plant Pathology*, 5(5):505–511.

Perring, T. M., Gruenhagen, N. M., and Farrar, C. A. (1999). Management of Plant Viral Diseases Through Chemical Control of Insect Vectors. *Annual Review of Entomology*, 44:457–481.

Picard, C., Rimbaud, L., Hendrikx, P., Soubeyrand, S., Jacquot, E., and Thébaud, G. (2017). PESO: a modelling framework to help improve management strategies for epidemics – application to sharka. *EPPO Bulletin*, 47(2):231–236.

Pickett, J. A. and Khan, Z. R. (2016). Plant volatile-mediated signalling and its application in agriculture: successes and challenges. *New Phytologist*, 212(4):856–870.

Rimbaud, L., Dallot, S., Borron, S., Soubeyrand, S., and Jacquot, E. (2015a). Assessing the Mismatch Between Incubation and Latent Periods for Vector-Borne Diseases : The Case of Sharka. *Phytopathology*, 105(11):1408–1416.

Rimbaud, L., Dallot, S., Bruchou, C., Thoyer, S., Jacquot, E., Soubeyrand, S., and Thébaud, G. (2019). Improving management strategies of plant diseases using sequential sensitivity analyses. *Phytopathology*, 109(7):1184–1197.

Rimbaud, L., Dallot, S., Gottwald, T., Decroocq, V., Jacquot, E., Soubeyrand, S., and Thébaud, G. (2015b). Sharka Epidemiology and Worldwide Management Strategies: Learning Lessons to Optimize Disease Control in Perennial Plants. *Annual Review of Phytopathology*, 53(1):357–378.

Robert, Y., Woodford, J. A. T., and Ducray-Bourdin, D. G. (2000). Some epidemiological approaches to the control of aphid-borne virus diseases in seed potato crops in northern Europe. *Virus Research*, 71(1-2):33–47.

Rousselin, A., Sauge, M. H., Jordan, M. O., Vercambre, G., Lescourret, F., and Bevacqua, D. (2016). Nitrogen and water supplies affect peach tree-green peach aphid interactions: the key role played by vegetative growth. *Agricultural and Forest Entomology*, 18(4):367–375.

Shaw, A. K., Peace, A., Power, A. G., and Bosque-Pérez, N. (2017). Vector population growth and condition-dependent movement drive the spread of plant pathogens. *Ecology*, 98(8):2145–2157.

Sisterson, M. S. and Stenger, D. C. (2013). Roguing with replacement in perennial crops: Conditions for successful disease management. *Phytopathology*, 103(2):117–128.

Sisterson, M. S. and Stenger, D. C. (2016). Disentangling effects of vector birth rate, mortality rate, and abundance on spread of plant pathogens. *Journal of economic entomology*, 109(2):487–501.

Smith, J. M. and Slatkin, M. (1972). The Stability of Predator-Prey Systems. *Ecology*, 54(2):384–391.

671 Tamburini, G., van Gils, S., Kos, M., van der Putten, W., and Marini, L.
672 (2018). Drought and soil fertility modify fertilization effects on aphid per-
673 formance in wheat. *Basic and Applied Ecology*, 30:23–31.

674 Thaler, J. S., Agrawal, A. A., and Rayko, H. (2010). Salicylate-mediated
675 interactions between pathogens and herbivores. *Ecology*, 91(4):1075–1082.

676 Van Emden, H. F. and Harrington, R. (2007). *Aphids as crop pests*. Cabi.

677 Zaffaroni, M., Cunniffe, N. J., and Bevacqua, D. (2020). An eco-physiological
678 model coupling plant growth and aphid population dynamics. *Journal of*
679 *The Royal Society Interface*, 17(172).

680 Zhao, X., Reitz, S. R., Yuan, H., Lei, Z., Paini, D. R., and Gao, Y. (2017).
681 Pesticide-mediated interspecific competition between local and invasive
682 thrips pests. *Scientific Reports*, 7(January):1–7.

683 Zust, T. and Agrawal, A. A. (2016). Mechanisms and evolution of plant
684 resistance to aphids. *Nature Plants*, 2(1):1–9.

685 # Figures and tables

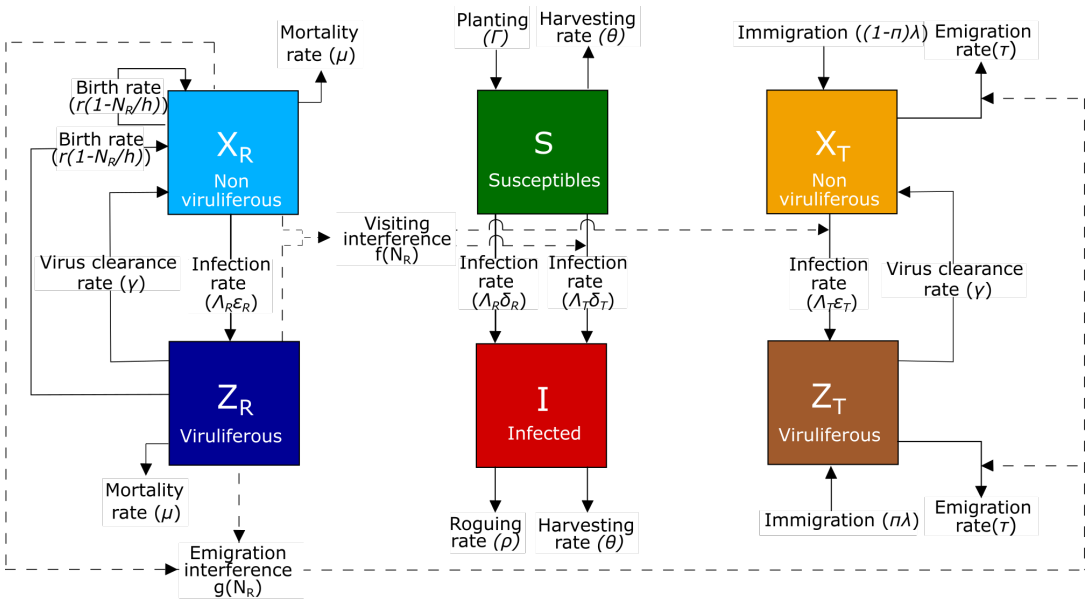

Figure 1: Schematic representation of the single host-multi vector model, where the the total number of host plants is partitioned into susceptible (S) and infected (I) individuals. Aphids are partitioned into non viruliferous (X_i) and viruliferous (Z_i), and are classified as resident ($i = R$) or transient ($i = T$). Dashed arrows identify the contacts between viruliferous aphids and susceptible plants, and between infected plants and non viruliferous aphids, which affect the infection rates. Circles identify the processes affected by inferences exerted by resident towards transient aphids (visiting interference in white and emigration interference in black). The total number of plants per hectare is $N_P = S + I$, the average number of resident aphids per plant is $N_R = X_R + Z_R$ and the average number of transient aphids visiting a plant per unit time is $N_T = X_T + Z_T$. Details on the processes involved are given in the main text.

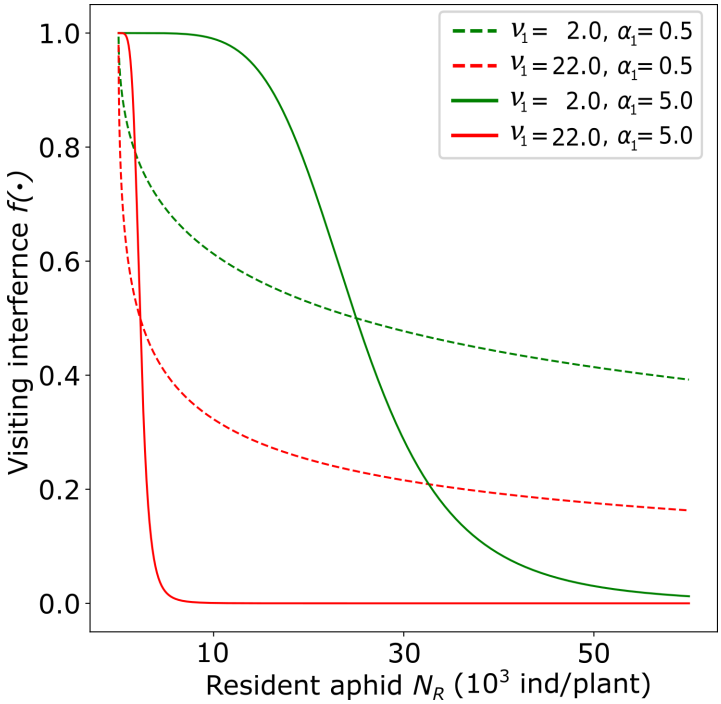

Figure 2: Proportionate decrease of the number of plants visited by a transient aphid as a function of the abundance of resident aphids, for two values of parameter ν_1 and α_1, in a plant displaying a reference plant hosting capacity (*i.e.* $h = h_R$, thus the value of $f(\cdot)$ is independent of the interference scenario).

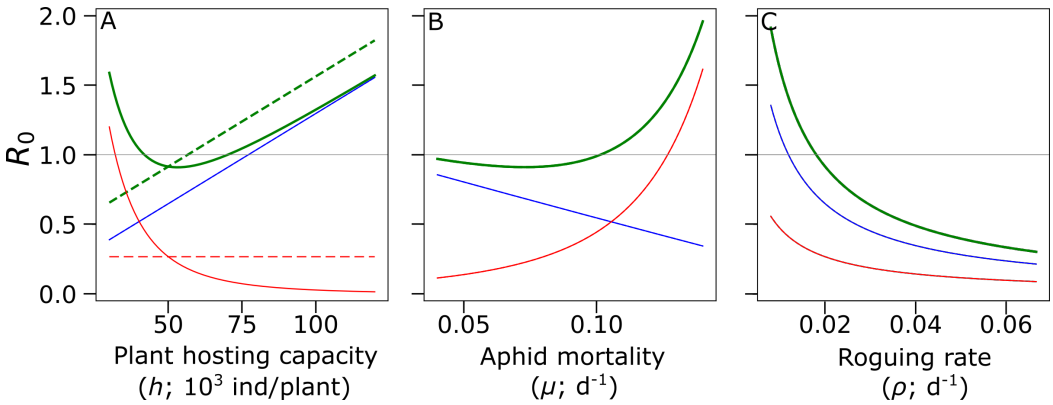

Figure 3: Response of the basic reproduction number R_0 (in bold and green) and its components R_0^R (in blue) and R_0^T (in red) to changes in (A) plant hosting capacity (h) under indirect (continuous line) and direct (dashed line) interference scenarios, (B) resident aphids mortality (μ), (C) roguing rate (ρ). Note that in (A) blue continuous and dashed lines overlap.

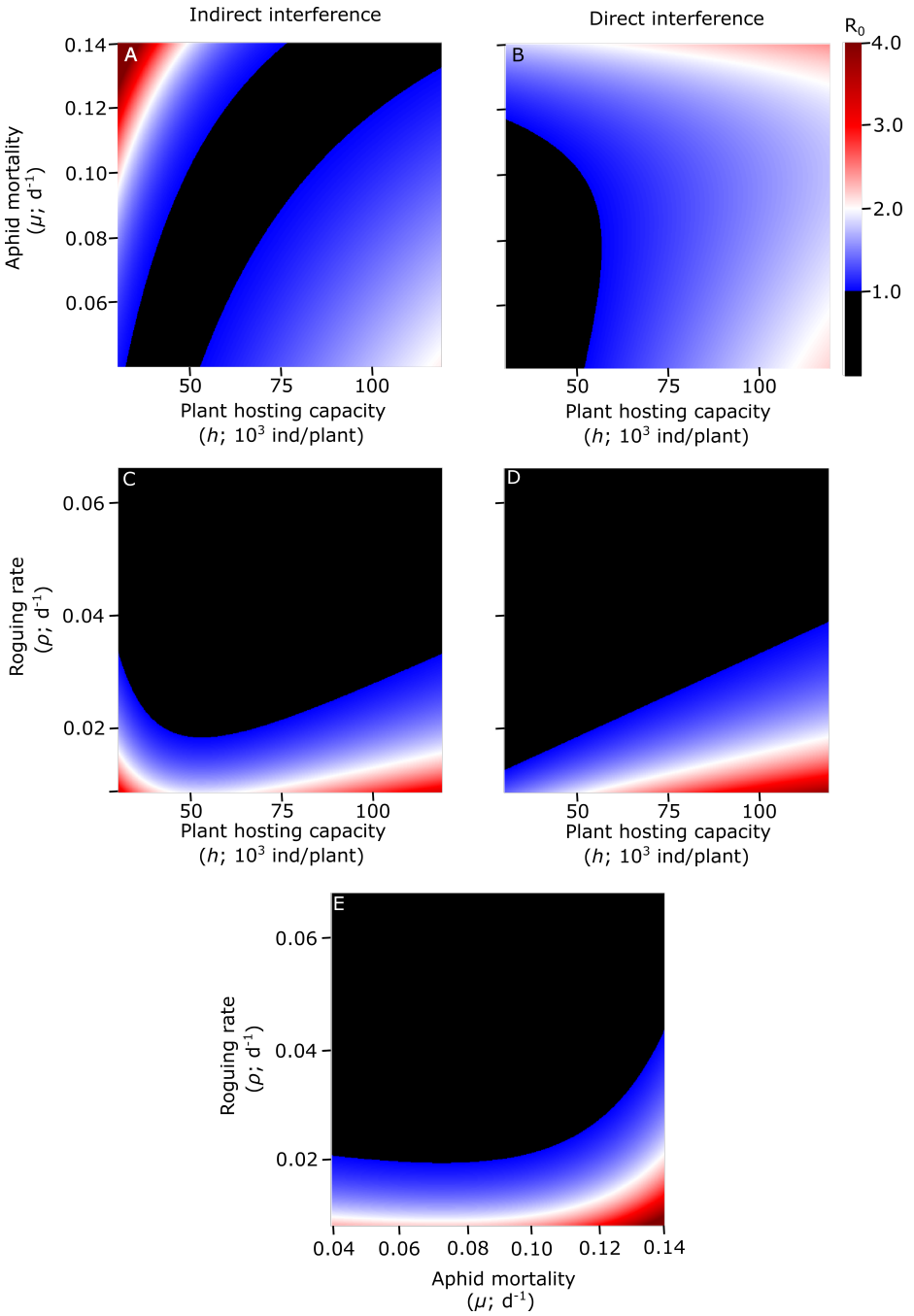

Figure 4: Response of R_0 to changes in: plant hosting capacity (h) and resident aphid mortality (μ) (A-B); plant hosting capacity (h) and roguing rate (ρ) (C-D); resident aphid mortality (μ) and roguing rate (ρ) (E), under different interference scenarios (indirect and direct). Note that the interference scenario has no effect on R_0 when μ and ρ are simultaneously varied (E). Black areas identify values of $R_0 < 1$, corresponding to disease eradication. Other model parameters are set to default values (Table 1).

Table 1: Model state variables and parameters

Sources: (1) Jeger et al. (1998); (2) Jeger et al. (2004); (3) Jeger et al. (2008); (4) Holt et al. (1997); (5) Madden et al. (2000); (6) Rimbaud et al. (2019); (7) Rousselin et al. (2016); (8) Fixed to an arbitrary, biologically-plausible reference value

Variable	Description	Dimensions		
S	Susceptible plants	plant ha^{-1}		
I	Infected plants	plant ha^{-1}		
X_R	Non viruliferous resident aphids	aphid plant^{-1}		
Z_R	Viruliferous resident aphids	aphid plant^{-1}		
X_T	Non viruliferous transient aphids	aphid plant^{-1}		
Z_T	Viruliferous transient aphids	aphid plant^{-1}		
Parameter	Description	Dimensions	Values	Source
Γ	Planting rate	plant day^{-1}	$\theta S + (\rho + \theta)I$	
Λ_R	Number of plants visited by a resident aphid	plant aphid^{-1} day^{-1}	0.05	(8)
Λ_T	Number of plants visited by a transient aphid	plant aphid^{-1} day^{-1}	8.5	(8)
δ_R	Probability of virus transmission from the resident aphid to the plant	dimensionless	0.04	(1)
δ_T	Probability of virus transmission from the transient aphid to the plant	dimensionless	0.04	(1)
ε_R	Probability of virus transmission from the plant to the resident aphid	dimensionless	0.02	(1)
ε_T	Probability of virus transmission from the plant to the transient aphid	dimensionless	0.02	(1)
α_1	Visiting interference curvature	dimensionless	1.00	(8)
ν_1	Visiting interference strength (for direct interference)	dimensionless	12.0	(8)
α_2	Emigration interference curvature	dimensionless	1.00	(8)
ν_2	Emigration interference strength (for direct interference)	dimensionless	12.0	(8)
ρ	Infected plant roguing rate	day^{-1}	0.02	(2,3)
θ	Plant harvesting rate	day^{-1}	0.003	(4)
r	Intrinsic growth rate of resident aphids	day^{-1}	0.21	(4)
h	Plant hosting capacity	aphid plant^{-1}	50,000	(7)
h_R	Reference plant hosting capacity	aphid plant^{-1}	50,000	(8)
μ	Mortality rate of resident aphids	day^{-1}	0.08	(8)
γ	Virus clearance rate in aphid vectors	day^{-1}	4	(1)
λ	Average number of transient aphids immigrating per plant	aphid plant^{-1} day^{-1}	$\tau T = 250$	derived
π	Fraction of viruliferous transient aphids entering the system	dimensionless	0	(8)
τ	Transient aphids emigration rate in absence of resident aphids	day^{-1}	0.5	(2,5)
N_P	Total number of plants	plant ha^{-1}	720	(6)
T	Average number of transient aphids per plant in absence of resident aphids	aphid plant^{-1}	500	(8)

Table 2: Summary of equilibrium behaviour. The value of state variables at the equilibrium are presented in the *Supplementary Information*.

Viruliferous aphids enter the system ($\pi > 0$)	Resident aphids are present ($\mu < r$)	Basic reproduction number (eq. 6)	$(\bar{S}, \bar{I}, \bar{X}_R, \bar{Z}_R, \bar{X}_T, \bar{Z}_T)$	Explanation
no	no	$R_0 < 1$	$(+, 0, 0, 0, +, 0)$	Transient aphids do not bear the disease from outside the system. Resident aphids are absent, the disease is spread by transient aphids but it does not persist in the system.
no	no	$R_0 > 1$	$(+, +, 0, 0, +, +)$	Transient aphids do not bear the disease from outside the system. Resident aphids are absent, the disease is spread by transient aphids.
no	yes	$R_0 < 1$	$(+, 0, +, 0, +, 0)$	Transient aphids do not bear the disease from outside the system. Resident and transient aphids spread the disease, but it does not persist in the system.
no	yes	$R_0 > 1$	$(+, +, +, +, +, +)$	Transient aphids do not bear the disease from outside the system. Resident and transient aphids spread the disease.
yes	no	- *	$(+, +, 0, 0, +, +)$	Transient aphids bear the disease from outside the system. Resident aphids are absent, transient aphids spread the disease.
yes	no	- *	$(+, +, +, +, +, +)$	Transient aphids bear the disease from outside the system. Resident and transient aphids spread the disease.

* The disease is always able to persist, regardless of whether the basic reproduction number is smaller or larger than 1.